Determinants of Blood Lead Concentrations to Age 5 Years in a Birth Cohort Study of Children Living in the Lead Smelting City of Port Pirie and Surrounding Areas

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ABSTRACT. Sources of variation and some principal determinants of blood lead concentration (PbB) were investigated in a cohort of children, followed to age 5 y, who were born near a lead smelter in Port Pirie, South Australia. The child's age and place of residence were the two variables most strongly predictive of PbB. A sharp increase in PbB occurred between 6 and 15 mo of age and was followed by a peak concentration that occurred at approximately 2 y of age, after which PbB steadily and consistently declined. Irrespective of age, the PbBs in children who lived in Port Pirie were significantly higher than levels identified in children who resided outside the city. There was no significant difference in PbB between boys and girls. Elevated PbB at each specific age was associated mainly with increased lead concentrations in the topsoil of the local residential area, employment of the father in the lead industry, parental smoking, and behaviors likely to cause ingestion of dirt. Blood samples taken from children at certain ages and during the warmer months contained more lead than samples obtained during the cooler months. The effects of these determinants on PbB during early childhood were basically consistent in both single and multivariable analyses.

THE ADVERSE EFFECTS of excessive lead exposure on the intellectual, cognitive, and behavioral development of young children have received increased atten-

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tion during the past decade.[1-3] Definitive evidence of neuropsychological effects that result from exposure to low levels of lead continues to be sought in prospective studies of child development. Some epidemiological studies have shown an inverse relationship between blood lead concentrations (PbBs) and early cognitive function,[4-7] and, therefore, the question of how exposure to environmental lead can be reduced in children has become a major public health concern.

Many researchers have described the relationship between PbBs and associated determinants.[8-10] Very limited longitudinal data on the variations and determinants of PbB during early childhood are available. Most data available have been derived from cross-sectional surveys of school-age children and adults or from clinically based groups of young children.

In 1979, the recruiting of pregnant women for a prospective study of lead exposure and its relationship to pregnancy outcome and early childhood growth and development was initiated in the South Australia town of Port Pirie (population 16 000), which is 200 km northwest of Adelaide. The town is located immediately downwind from a large and longstanding lead-smelting facility. Although emissions are now controlled tightly, past activities of the smelter, the use of lead-bearing waste as landfill, and unloading activities at the railhead have left the city with a legacy of extensive environmental contamination with lead, much of it finely particulate and sufficiently mobile to be a problem in dust and topsoil.

The Port Pirie Cohort study is concerned primarily with early childhood physical and neurobehavioral development and the relationship of cumulative lead exposure with each. The study, however, has highlighted the relationship between PbB and socioeconomic status, behavioral factors, and environmental circumstances. Determinants of PbBs in the same cohort have already been described for children who are 2 y of age or younger[11]; this paper, therefore, extends the findings to age 5 y, i.e., just prior to attending school.

Materials and methods

Study population. The children in the cohort were born in the town of Port Pirie or in the surrounding rural area within a radius of approximately 30 km; the rural area included the townships of Laura, Crystal Brook, Port Broughton, and Gladstone. A total of 831 pregnant women were enrolled in the study from May 1979 to May 1981. This number accounted for an estimated 90% of all new pregnancies during this period. Seven hundred twenty-three of the 831 pregnancies were followed to single live births. The numbers of children who remained in the cohort were 646, 607, 585, 556, 530, and 513, and their ages were 6, 15, 24, 36, 48, and 60 mo, respectively. The majority (i.e., approximately 80%) of the children lost to follow up during the 5 y of postnatal study were in families that left the Port Pirie district; a few families discontinued participation. Additional information about the sociodemographic characteristics of the children who remained in the cohort and about those lost to follow up is available elsewhere.[12]

Data collection. Capillary blood samples for measurement of PbBs were obtained under standardized conditions and rigorous procedures.[13] Samples were collected from each child at 6, 15, 24, 36, 48, and 60 mo of age. In a separate validation study conducted in a group of 47 Adelaide metropolitan children aged 2–4 y, a close correlation ($r = .97$) was observed between lead concentrations obtained with capillary sampling and with simultaneous venous sampling.[14] At the same time blood samples were collected, the nurse-interviewer also conducted a structured interview and obtained information on a range of demographic, behavioral, and environmental factors.

Laboratory analysis. Measurements of PbBs were performed in the Department of Chemical Pathology at the Adelaide Centre for Women's and Children's Health (formerly the Adelaide Children's Hospital). Blood lead concentrations were measured by electrothermal atomization atomic absorption spectrometry.[15] The blood lead assay performed in this cohort study was described previously and was subject to internal and external quality control procedures, and satisfactory results were obtained consistently. A certified commercially prepared product was used to monitor intra-batch accuracy and to ensure uniformity between batches. External quality control, which entailed assay of regularly supplied samples, was maintained by the Standards Association of Australia and the international programs operated by the Health Department of Pennsylvania (USA) and the Wolfson Research Laboratories (Birmingham, UK). Individual PbBs were adjusted to a packed cell volume of 35%.

Soil and dust lead. Because the dust-hand-mouth route may be a predominant form of lead exposure during early childhood, the relationship between PbB and three local measures of dust in soil was examined. (1) A survey of lead in topsoil was conducted by the South Australian Health Commission;[16] (2) lead in ceiling dust was surveyed by the Department of Environment and Planning[17] (ceiling dust was chosen because many houses are designed with an airflow through the roof space, and dusts collected between the ceiling rafters are generally representative of dusts deposited during the life of each house); and (3) lead in dust on window-sills[17] was surveyed by the Department of Environment and Planning.

In each survey, soil- or dust-lead values were averaged for 200-m^2, an area that was defined by a rectangular grid drawn across the residential area of Port Pirie. The average for the appropriate area was then used as a covariate for all children who resided in that area. All three measures were strongly predictive of a child's PbB, but the Health Commission data were more extensive than the others and were, therefore, chosen for all subsequent analyses (Fig. 1). It should be noted that sites sampled by the Health Commission were not random, but focused on homes of children who had unacceptably high PbBs. Thus, the reported concentrations may be overestimates of the general situation within Port Pirie, but the volume of data collected in this manner would have insured that the final summary data closely approximated the true situation, and Figure 1 is

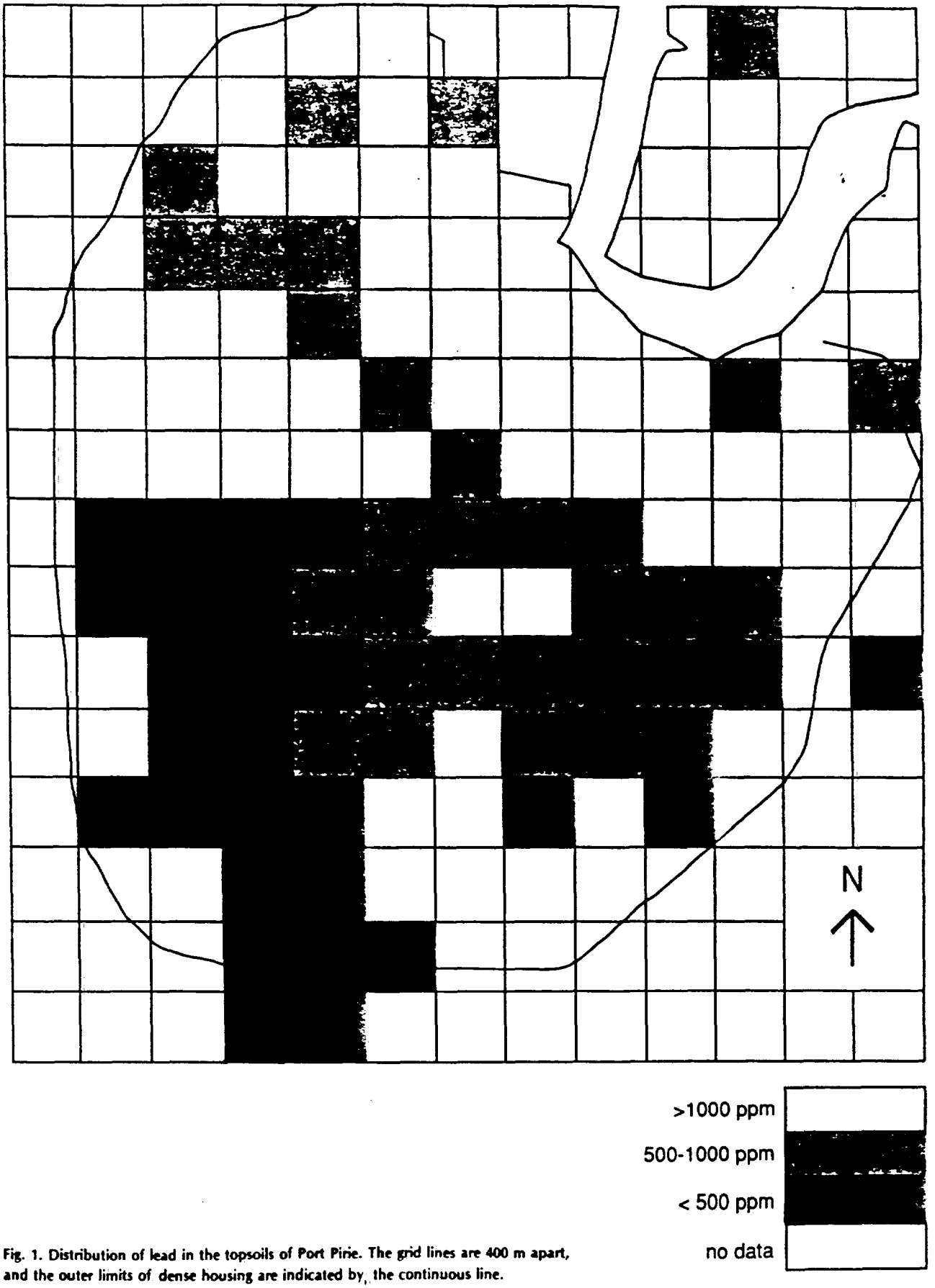

Fig. 1. Distribution of lead in the topsoils of Port Pirie. The grid lines are 400 m apart, and the outer limits of dense housing are indicated by the continuous line.

remarkably similar to the maps published by the Department of Environmental Planning.[17] Nevertheless, it was prudent to use the soil data in a categorized form, which avoided assumptions of curvilinearity and allowed inclusion of the children who lived outside Port Pirie and for whom there were no soil lead data.

Analysis of lead concentrations in the samples collected by the Health Commission was conducted by flame atomic absorption spectrometry, for which the ethylenediamine tetraacetic acid (EDTA) extraction method was used.[18]

Other variables. Social status was quantified by a scale proposed by Daniel[19] and was based on the perceived prestige of the father's occupation. Paternal employment at the smelter was used as a separate variable. Parental smoking was defined as the number of parents who smoked (i.e., 0, 1, or 2). Behaviors likely to lead to ingestion of dirt included mouthing activity, eating dirt, and/or sucking fingers. Each behavior was categorized by reported frequency, i.e., often, a little, or never. Education was estimated as the number of years the child's father/mother had attended secondary school.

In the town of Port Pirie and its surrounding rural area, some families collect rainwater runoff from rooftops and store it in tanks for drinking, cooking, or other domestic uses. This supply can be contaminated by lead-containing dust; therefore, use of rainwater was employed as one of the variables that might influence a child's lead burden.

Because exposure to environmental lead may vary during the seasons, a simple variable to indicate whether each child's birthday occurred in the warmer or cooler months of the year was also included. April–October were considered cooler months, and November–March were the warmer months.

Statistical analysis. Because blood lead concentrations are typically skewed, analysis was performed on the natural logarithm of PbB, and all means reported are geometric and not arithmetic. Multiple regression analyses were conducted in an attempt to construct an appropriate model to explain the variation in PbBs. Residual analysis supported the regression model assumptions of normality.

The final results of the multiple regression analysis were reported as percentage differences. For example, if the estimate of the regression coefficient (of log PbB) for girls was −.057 relative to a value of 0 for boys, this would be equivalent to concluding that girls have a PbB that is 100[exp(−.057) − 1] = −5.6% "above" (i.e., 5.6% below) that of their male counterparts, assuming values for all other independent variables remain the same.

Results

Variations in geometric mean PbB with respect to age, gender, and location of residence are indicated in Figure 2. There was a considerable increase in PbB between the ages of 6 and 15 mo, and the peak concentration observed (Port Pirie, 25.1 μg/dl; non-Port Pirie, 17.4 μg/dl [conversion factor for SI units: 1.0 μmol/l = 20.7 μg/dl]) occurred at 2 y of age and was followed by a subsequent slow and steady decline at later ages. However, the mean PbB in children aged 5 y (Port Pirie, 18.6 μg/dl; non-Port Pirie, 11.5 μg/dl) remained significantly higher than the levels recorded at birth (Port Pirie, 9.4 μg/dl; non-Port Pirie, 5.7 μg/dl).

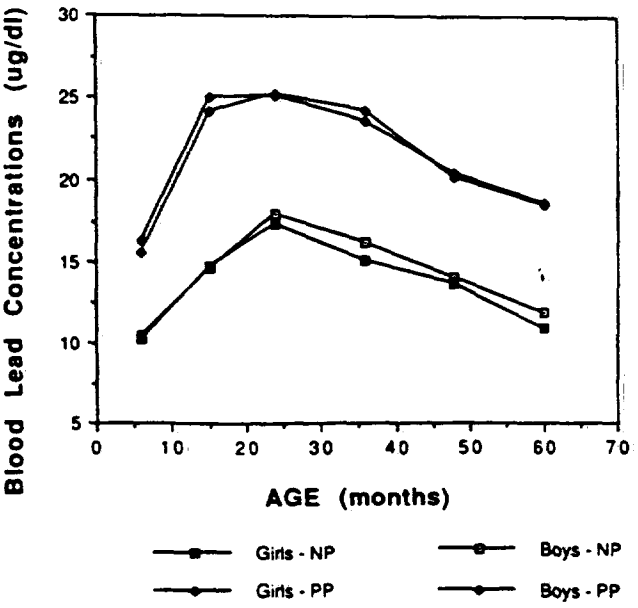

Fig. 2. Geometric mean blood lead concentration, by age, sex, and location of residence. (PP = Port Pirie; NP = outside Port Pirie).

The PbBs in children who lived in Port Pirie were considered higher (53.8%–68.5%) at all ages than in children who resided outside the city; the maximum difference occurred at 15 mo of age. At no age was there a statistically significant difference between boys and girls.

There was an inverse gradient in geometric mean PbB (Table 1) as social status increased in each age group ($p < .01$). The PbBs in children whose parent(s) smoked tended to be 1.7–3.0 μg/dl (10%–19%) higher than in children whose parents did not smoke. Mouthing activities, eating dirt, and sucking fingers were associated with elevated PbBs.

There was a strong relationship between a child's PbB and the father's employer. The PbBs of children whose fathers were employed in the smelter (17.2–25.4 μg/dl) were significantly higher than those of children whose fathers were employed elsewhere (10.6–20.9 μg/dl). At each age there was a clear, positive gradient in mean PbB (11.1–28.3 μg/dl), relative to local soil lead concentrations. The gradients were stronger at age 6–15 mo than at all subsequent ages. Each age-specific gradient in mean PbB "relative to," including lead-exposure employment of parent and topsoil lead levels, was highly statistically significant ($p < .01$). At each age the mean PbB was higher during the warmer months and lower in cooler months; this difference was statistically significant at ages 6 mo ($p = .057$), 15 mo ($p = .001$), 36 mo ($p = .005$), and 48 mo ($p = .036$).

The results of multiple regression analysis (Table 2) showed that the associations between PbBs and topsoil lead levels, employment at the smelter, parental smoking, behaviors likely to cause ingestion of dirt, and season remained statistically significant. Nevertheless, apparent effects of gender, work duration of mother, parental secondary education and occupation, and rainwater use on mean PbB were not found in this multiple regression model.

Discussion

The results indicated that there was a significant increase in either exposure to, or uptake of environmental lead in infancy. The peak mean PbB appeared to occur at age 2 y, and there was a subsequent slight but steady decline thereafter. These findings corroborate the reports from several other community-based studies conducted in the United States and Europe, each of which showed a peak in mean PbB at 2–3 y of age.[20,21] Although the Second National Health and Nutrition Examination Survey in the United States[22] and a few other studies[23,24] did not report a peak in mean PbB at this age exactly, it is apparent that the reported values in infancy were considerably higher than those recorded by their maternal age group and that a rise in PbB must have occurred soon after birth, when cord blood lead is quantitatively similar to maternal PbB. A definitive explanation for this phenomenon remains elusive, but it is likely to involve an enhanced ability to absorb lead and/or an increased ingestion of lead that

Table 1.—Variations in Blood Lead Concentrations, by Gender, Season, Socioeconomic Status, Parental Smoking, Behaviors, Workplace of Father, and Topsoil Lead Levels

Factors	Age (mo) 6 (*n* = 646)	15 (*n* = 607)	24 (*n* = 585)	36 (*n* = 556)	48 (*n* = 530)	60 (*n* = 513)
Gender						
Male	14.0 (1.02)	20.4 (1.02)	21.4 (1.02)	19.6 (1.02)	16.4 (1.02)	14.6 (1.02)
Female	14.5 (1.02)	21.1 (1.02)	20.8 (1.02)	19.1 (1.02)	16.4 (1.02)	14.3 (1.03)
Season		*		*	†	
Warmer	14.8 (1.02)	22.1 (1.02)	21.3 (1.02)	20.2 (1.02)	17.0 (1.03)	14.7 (1.03)
Cooler	13.9 (1.02)	19.9 (1.02)	21.0 (1.02)	18.7 (1.02)	16.0 (1.02)	14.2 (1.02)
Socioeconomic status	*	*	*	*	*	*
Lowest	17.2 (1.07)	24.0 (1.08)	24.7 (1.08)	21.9 (1.08)	19.5 (1.07)	18.1 (1.07)
Low	16.6 (1.03)	25.2 (1.03)	25.5 (1.03)	23.0 (1.03)	19.6 (1.03)	17.6 (1.03)
Middle	14.2 (1.04)	19.8 (1.04)	19.8 (1.03)	19.1 (1.03)	16.2 (1.03)	14.0 (1.03)
High	13.2 (1.03)	19.3 (1.03)	19.8 (1.03)	17.9 (1.03)	15.2 (1.03)	13.2 (1.04)
Highest	12.3 (1.04)	18.2 (1.04)	18.7 (1.04)	16.2 (1.04)	13.7 (1.04)	12.0 (1.04)
Parental smoking	*	*	*	*	*	*
None	13.5 (1.03)	19.0 (1.03)	19.4 (1.02)	17.8 (1.02)	15.1 (1.03)	13.4 (1.03)
One	14.3 (1.03)	21.1 (1.03)	21.5 (1.02)	19.9 (1.03)	16.8 (1.03)	14.8 (1.03)
Both	15.0 (1.03)	22.5 (1.03)	23.1 (1.03)	21.1 (1.03)	18.4 (1.03)	16.4 (1.04)
Mouthing activity			*	*	*	*
No	–	20.5 (1.06)	19.9 (1.02)	18.6 (1.02)	16.0 (1.02)	14.2 (1.02)
Yes	–	20.8 (1.02)	22.0 (1.02)	21.0 (1.03)	18.2 (1.04)	15.8 (1.06)
Dirt eating			†	*	*	
No	–	20.2 (1.04)	20.6 (1.02)	18.8 (1.02)	16.3 (1.02)	14.4 (1.02)
Yes	–	20.9 (1.02)	21.9 (1.02)	22.4 (1.04)	18.6 (1.06)	14.9 (1.08)
Finger sucking			†	†	†	*
Never	–	20.8 (1.02)	20.6 (1.02)	18.8 (1.02)	16.1 (1.02)	14.0 (1.02)
Occasionally	–	21.2 (1.03)	22.1 (1.03)	20.6 (1.03)	16.8 (1.03)	15.3 (1.03)
Often	–	19.3 (1.06)	22.2 (1.08)	19.3 (1.05)	17.6 (1.07)	15.5 (1.10)
Father's job	*	*	*	*	*	*
Non-Port Pirie	10.6 (1.03)	15.4 (1.03)	16.4 (1.03)	15.3 (1.02)	12.9 (1.03)	11.1 (1.03)
Nonsmelter	14.5 (1.02)	20.9 (1.03)	20.8 (1.03)	19.4 (1.03)	17.2 (1.03)	14.8 (1.03)
Smelter	17.2 (1.02)	25.3 (1.02)	25.4 (1.02)	23.2 (1.02)	19.3 (1.02)	17.7 (1.02)
Soil lead (ppm)	*	*	*	*	*	*
Non-Port Pirie	11.1 (1.03)	15.5 (1.03)	16.9 (1.03)	15.1 (1.02)	13.0 (1.03)	11.1 (1.03)
< 500	14.7 (1.03)	22.0 (1.03)	22.2 (1.03)	21.2 (1.03)	17.7 (1.03)	16.5 (1.03)
<1 000	15.8 (1.03)	23.5 (1.03)	23.3 (1.03)	21.7 (1.03)	18.4 (1.03)	16.5 (1.03)
≥ 1 000	18.7 (1.03)	28.3 (1.03)	26.7 (1.03)	25.1 (1.03)	22.0 (1.03)	18.8 (1.04)
No data‡	13.3 (1.10)	20.8 (1.11)	19.9 (1.08)	19.8 (1.09)	15.4 (1.13)	13.0 (1.17)

Note: Geometric standard errors appear within parentheses.
*$p < .01$.
†$p < .05$.
‡Number of children for whom lead data were unavailable are 17, 20, 20, 16, 13, and 10 at the ages of 6, 15, 24, 36, 48, and 60 mo, respectively.

Table 2.—Relative Shifts (%) of Blood Lead Concentrations, Estimated by Multiple Regression Analyses

Factors (reference) categories)	Categories	Age (mo) 6	15	24	36	48	60
Demography							
Gender (males)	Females	−2.6	1.9	−4.3	−4.8	1.4	−5.6
Secondary education (y)							
Father (⩽ 3)	> 3	7.3	−4.6	−5.8	−4.7	−5.2	−6.2
Mother (⩽ 3)	> 3	3.7	−1.1	0.2	−2.0	−1.9	4.1
Socioeconomic status							
(Lowest)	Lower	−0.3	2.1	−7.5	−11.2	−10.3	2.0
	Middle	−0.3	−4.4	−21.3*	−16.3	−11.3	−8.6
	Higher	−8.7	−1.0	−17.0	−16.0	−13.6	−8.2
	Highest	−13.5	−6.6	−20.3*	−22.7*	−19.6	−13.4
Occupation							
Mother (lower)	Middle	−10.8	1.5	3.7	−6.7	5.4	−1.1
	Higher	−0.4	0.8	−2.0	−7.9	−3.1	−10.2
Father (lower)	Middle	1.1	−6.2	1.3	0.4	−2.2	1.9
	Higher	−3.4	−7.5	−0.9	0.1	−1.4	−1.2
Mother's working time							
(No job)	Short	−12.0	3.4	0.3	0.4	3.0	−3.4
	Middle	27.6	−1.3	−4.4	0.5	−5.2	−4.7
	Long	−2.0	5.1	−3.2	3.7	−1.6	−7.6
Behavior							
Mouthing activity (no)	Yes	—	4.7	9.0*	3.5	2.5	−0.1
Dirt eating (no)	Yes	—	10.9*	−3.2	9.9	8.9	−11.8
Finger sucking (no)	Sometimes	—	−5.1	8.8†	1.3	0.7	6.6
	Frequently	—	2.4	8.4	7.3	15.6*	16.7*
Environment							
Season (warmer)	Cooler	−4.6	−10.1*	−4.2	−7.5*	−7.9*	−6.2
Father's employment							
(Non-Port Pirie)	Nonsmelter	26.1†	14.9*	17.4†	7.8	19.9†	10.3
	Smelter	52.2†	29.7†	29.4†	21.7†	28.3†	23.1†
Parental smoking							
(None)	One	3.6	5.8	7.4*	5.9	8.2*	4.9
	Both	10.3	9.6	12.3†	9.6*	17.4†	11.0*
Rainwater use							
Cooking (never)	Sometimes	−15.0	−6.2	1.8	−3.2	4.3	−1.2
	Frequently	−4.5	−5.9	0.8	−5.0	2.7	−3.5
Drinking (never)	Sometimes	14.6	7.5	3.0	7.5	−10.2	8.1
	Frequently	13.0	−2.1	−5.4	−3.9	−7.4	−4.6
Soil lead levels							
(Non-Port Pirie)	Lower	5.2	13.0*	2.4	13.0*	7.5	20.8†
	Middle	18.6†	29.3†	13.3†	23.5†	18.4†	24.7†
	Higher	43.3†	47.7†	25.0†	36.3†	32.3†	36.2†

*$p < .05$.
†$p < .01$.

results from various mouthing activities during early childhood development. Clinical studies have shown, with certainty, that approximately 40–50% of ingested lead is absorbed from the gastrointestinal tract during early childhood, whereas adults absorb only 5–10% in this manner.[25]

Mean PbBs decreased significantly during ages 2–5 y. This probably was not a consequence of secular changes in the environment because a decrease has been generally observed in many of the studies mentioned; also, there was limited evidence that a cohort effect was operating within Port Pirie when PbB-by-age profiles were constructed for each of the 3 y of recruitment (1979 to 1982). A more feasible explanation is in terms of those factors already invoked to explain the initial rise in PbB, e.g., decreased hand-to-mouth activity (associated with normal child development); this decrease in activity leads to reduced ingestion of lead and/or a decrease in ability over time for the child to absorb ingested lead.

The lack of a difference in mean PbB between preschool girls and boys accords with other studies.[26,27] Divergence of PbBs for boys from girls usually occurs at later ages (6 y to adolescence), and boys consistently record higher values for mean PbB than girls.[22,28] The definite mechanism for this occurrence remains unclear. One explanation is the greater opportunity for boys to ingest lead from their environment because they usually play outside more often than do girls. Moreover, the blood lead measurements were not ad-

justed by packed cell volume in these studies, and the differences could, therefore, be explained in part by the presence of a higher red cell or hemoglobin concentrations in the boys during and after puberty.

The results of analysis indicate that two variables are particularly conspicuous in their association with PbBs. There is a strong, positive gradient in percentage shifts of PbBs relative to local topsoil lead levels. The highest concentrations in PbB have been observed in children who lived in areas with the highest topsoil lead content. This dose-response relationship is evidence for a direct effect of the lead, either in the topsoil or in the air, on body lead burden. It has been predicted elsewhere[29] that the estimated mean natural log-transformed PbB could increase by 0.231 μg/dl for each unit increase in natural log of the soil lead level.[29] Evidence that lead in household and play yard dust is a major source of childhood body lead burden has been adduced by other observers.[29,30]

Employment of parents at the smelter was another variable significantly associated with higher PbB. Since 1984–1985, employees have been provided with work clothes that do not have to be taken home and with showers. However, these changes occurred too late for their impact on PbBs to become apparent in this study. The persistence of this finding in the multiple regression context suggests that there is no strong confounding with other variables, e.g., place of residence or social status.

There was a positive association between PbB in young children and parental smoking status, a finding that is in agreement with other studies. Sherlock et al.[31] reported higher PbBs in children whose parent(s) smoked, compared with children who resided with nonsmoking parents, although the difference was not statistically significant. Furthermore, in a study of environmental exposure to lead and arsenic among children who lived near a glassworks, there were indications that parental smoking habits had a significant effect on PbB in children.[32] Recently, Willers et al.[33] reported a significant association between higher PbBs in children and parental smoking, and the author ascribed this result to the difference in home environment, family lifestyle, and small airways diseases that affect absorption of inhaled lead particles.

It is difficult to conduct a quantitative assessment of children's behaviors, and the anticipated effects of behavior on PbB have not, therefore, been observed in this study. However, there were some indications that PbBs in children are related to their behaviors, and statistical significance was observed at some ages. The combination of mouthing activities, eating dirt, and sucking fingers supports the study by Charney et al.,[34] who found that a group of 50 children who had high PbBs (40–79 μg/dl) played in outside soil, mouthed objects, and sucked their fingers more often than a matched group (PbB, ≤ 29 μg/dl).

The mean PbB was influenced by the month in which the children were studied, i.e., peak in PbB during the summer, a finding that has been described by others in nontropical climates.[35] One possible explanation is the seasonal variation in the amount of lead exposure; most children spend more time playing outside in the summer than in winter, and the rain in winter may stabilize dust.

The significant gradients in mean PbB in relation to mother's education, parental occupation, and use of rain water were no longer statistically significant in multiple regression analysis. This suggests that these variables were at least partially confounded with other factors. For example, the association between the PbB and use of rain water was obviously confounded by residential area. Outside Port Pirie, two-thirds of the children always use rain water for drinking and in prepared foods, whereas one-third of the children used little or no rain water at all. However, the situation was exactly the reverse in Port Pirie.

It is cautioned, however, that the assessment of variation in PbB depends on an understanding of the environmental sources of lead and the means by which lead enters the body. Young children are particularly likely to have many sources of lead exposure from air, food paint, dust, and dirt. The use of a 16-variable model explained 36.3% (at 36 mo) or less (at other ages) of the variation in PbB during early childhood, and other factors associated with changes in PbB almost certainly remain to be identified. Although many of the houses in Port Pirie are more than 50 y old and contain leaded paints, scanning electron microscopy of samples of household dusts[36] revealed that the absolute quantity of paint flakes present in the dust samples was very low and that the total lead levels in the dust were not correlated with measured paint contamination. Moreover, no correlation between lead-based paints and PbBs of children was observed, and Body[36] concluded that unless the individual child exhibited pica, leaded paints were unlikely to contribute to elevated PbBs.[37] Hence, incorporation of paint data in this analysis is unlikely to explain significantly more variation in PbB. Subsequent to the smelter's commission of a 205-m stack in 1979 (just prior to the first recruitment of pregnant women for this study), fugitive lead emissions from the smelter have been low.[38] The general level of total suspended particulate lead in Port Pirie was approximately 0.43 μg/m³ in 1984, which is well below the Australian National Health and Medical Research Council's criterion that "the maximum permissible level of lead in air in the urban environment should be 1.5 micrograms per cubic metre averaged over 3 calendar months." Data on airborne lead were not sufficiently widespread to enable construction of contours of average exposure throughout the residential area for inclusion in this analysis.

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